

This was incised and drained under local anaesthetic and the pus cultured grew *P aeruginosa* sensitive to ciprofloxacin.

After 11 weeks' treatment with ciprofloxacin, the skin between the abscesses broke down to reveal a cavity; betadine packs were inserted and necrotic tissue at the wound edges was debrided. After 14 weeks' treatment with ciprofloxacin and abscess cavity packing, swabs taken from the cavity yielded no bacterial growth. Wound care with granu-flex was commenced and treatment with ciprofloxacin continued for a further 5 weeks. The abscesses were completely healed 23 weeks after the patient's initial presentation.

Breast abscesses are common in women but rare in men. To our knowledge there is only one published report of non-lactational breast abscesses in an HIV-positive individual, a woman whose breast abscess was caused by *Mycobacterium tuberculosis*.¹ We believe that ours is the first reported case of breast abscesses in an HIV positive male. Recently, a case of gonococcal mastitis has been reported in a homosexual male who wore a nipple ring, but the HIV status of this patient was not known.²

Periareolar breast abscesses are associated with underlying periductal mastitis³ and heavy cigarette smoking has also been implicated in their aetiology.^{4,5} It is interesting to note that our patient smoked approximately 30 cigarettes a day. Culture of *P aeruginosa* in this case is in keeping with the increased susceptibility to infection with this organism seen in immunosuppressed patients.

Some authors have recommended that successful management of periareolar abscesses necessitates surgical excision of infected tissue, including partial nipple excision where necessary.^{6,7} One of these studies⁶ showed a higher risk of relapse in those cases managed by simple incision and drainage. However, none of these studies related to HIV positive patients. The patient we describe was generally well throughout this period and despite a very low CD4+ count recovered with outpatient treatment. Subsequently, despite a general deterioration in his health in association with a further fall in his CD4+ lymphocyte count, there has been no recurrence of the abscesses.

S P HIGGINS*
Y F STEDMAN*
N J BUNDRED†
P D WOOLLEY*
P CHANDLER*

Departments of Genitourinary Medicine* and Surgery,†
University Hospital of South Manchester,
Manchester M20 8LR, UK

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Lactic acidosis, non-Hodgkins lymphoma and the acquired immunodeficiency syndrome

Chattha *et al* have recently reported seven cases of patients with the Acquired Immunodeficiency Syndrome (AIDS) who developed severe lactic acidosis in the absence of hypoxaemia, malignancy or other obvious causes.¹ We report a case of AIDS related Burkitts lymphoma complicated by Type B lactic acidosis.

A 26 year old male, diagnosed as Human Immunodeficiency Virus (HIV) antibody positive in 1985 secondary to intravenous drug use, was admitted in November 1992 with a 3 week history of generalised malaise, anorexia, and subjective weight loss. He had a past history of exposure to hepatitis B and C viruses as a result of his drug use. He has not used non-prescribed drugs since the time of diagnosis in 1985, and had not received zidovudine therapy at any stage.

On admission he was afebrile and normotensive. There were no clinical stigmata of chronic liver disease. Abdominal examination revealed splenomegaly and an epigastric mass. Respiratory examination revealed left basal coarse crepitations. Sputum culture grew *Haemophilus influenzae* and *Streptococcal pneumoniae* species but at no stage was the patient clinically septic or shocked. A chest radiograph was normal. Initial investigations pH = 7.23 (7.35-7.45), pO₂ 9.00 kPa (11.00-15.00), pCO₂ 5.46 kPa (4.50-6.00), oxygen saturation 91%, actual bicarbonate 16.0 mmol/l (normal 22-28), standard bicarbonate 16.0 mmol/l (normal 21-27) and base excess 10.0 mmol/l (normal -2.0-2.0). Full blood count was normal. Serum biochemistry showed a urea of 13 mmol/l (normal 3-7) and creatinine of 75 µmol/l (normal 50-125). Urate level was 861 mmol/l (normal 100-450). Liver function tests showed elevation and with some changes from assays in 1989 (table). Lactate dehydrogenase (LDH) level was 1081 IU/l (100-350). The anion gap was calculated at 23 (normal 12-15). Blood glucose was 3.5 mmol/l and toxicology screen for illicit drug use was negative. Lactate levels were recorded elevated at 7.9 mmol/l (normal 0.5-2.2). All other causes of elevated anion gap metabolic acidosis were excluded by appropriate laboratory measures.

Repeat LDH was 3696 IU/l, the subfractionation of which showed predominant LD2 and LD3 isoenzymes, suggestive of lymphoproliferative or myeloproliferative origin. Abdominal ultrasound confirmed liver

Table Liver function test findings

	(Normal)	1989	Apr 1992	Nov 1992
Alkaline phosphatase	(30–100 IU/l)	93	149	187
T Protein	(60–80 g/l)	74	75	75
Albumin	(35–50 g/l)	47	40	35
Bilirubin	(0–17 µmol/l)	9	8	6
Gamma GT	(10–55 IU/l)	137	158	156
AST	(7–40 IU/l)	55	98	93
LDH	(100–350 iu/l)	204	210	1081

parenchyma of equal echogenicity, splenomegaly and a lobulated mass extending from the porta hepatis to the epigastrium.

Bone marrow biopsy diagnosed Burkitt's lymphoma. He was treated with standard combination chemotherapy (CD4 count = $190 \times 10^6/l$). Follow-up lactate levels on day 6 were 12.2 mmol/l. Twenty four hours post chemotherapy the LDH level rose to 8,019 IU/l, with LD2 and LD3 predominance, in association with deteriorating renal function with hyperuricaemia and hyperphosphataemia. This heralded the development of tumour lysis syndrome. Lactate levels were 7.9 mmol/l. The patient remained acidotic with a pH ranging from 7.29–7.34 despite chemotherapy in combination with bicarbonate infusions. He died 48 hours after diagnosis, seven days after presentation.

Patients with HIV infection may develop type A lactic acidosis associated with tissue hypoxia secondary to sepsis or shock, as occurs in other population groups. As recently reported they can also develop lactic acidosis in the absence of hypoxaemia or another cause.¹ Type B lactic acidosis occurs in association with leukaemia or lymphoma.

The pathogenesis of malignancy related type B lactic acidosis has been attributed to overproduction of lactic acid by the tumour cells as shown by in vitro studies of Burkitt's malignant cells. Alternatively it may occur as a consequence of reduced hepatic clearance in cases of hepatic metastatic disease. In this case report, although there were elevated liver enzymes, these were most probably related to the patient's past exposure to hepatitis B and C viruses and the alteration seen in these enzymes over the 3 year period were probably a consequence of chronic hepatic disease progression. Furthermore abdominal ultrasonography showed no evidence of metastatic disease.

Another interesting facet of this case concerns the abnormalities observed in regard to LDH analysis. The activity of this glycolytic enzyme is present in plasma as five distinct

isoenzymes, LDH 1–5, and analysis of both plasma total LDH and LDH fractions is still widely used in the investigation of many disease processes including lymphoproliferative neoplasia such as leukaemia and non-Hodgkin's lymphoma. In relation to the HIV positive population elevated LDH is a known accompaniment of *Pneumocystis carinii* pneumonia where it has both diagnostic and prognostic potential.² However, fractionation of these LDH levels has revealed an isomorphous pattern³ which has no diagnostic specificity. More recently Pugin *et al*⁴ have reported profound elevations in LDH in AIDS related pulmonary toxoplasmosis but isoenzyme changes were not recorded.

In this case the elevation in LDH was induced by a Burkitt's lymphoma for which HIV infection provides a significantly increased risk. Furthermore a predominant LD2, LD3 isoenzyme pattern was noted throughout the disease period and an increased incidence of this same zymogram has been documented in lymphoproliferative malignancies.^{5,6} Thus this case report confirms an alternate cause for elevated LDH in HIV positive patients and shows that subfractionation techniques can help differentiate between causes. In particular LD2 and LD3 isoenzyme pattern may be indicative of an underlying lymphoma. We suggest that such methods should be incorporated in the investigation of all HIV positive patients with elevated serum LDH levels.

C BERGIN
R PILKINGTON
C MCCREARY
F MULCAHY

Department of Genitourinary Medicine,
St James' Hospital, James's Street,
PO Box 580, Dublin-8

V CROWLEY
Department of Clinical Biochemistry,
St James' Hospital, Dublin

Address correspondence to: Dr C Bergin
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